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Renal dysfunction in a patient with a Roux-en-Y gastric bypass surgery

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CLINICAL PRESENTATION

A 69-year-old man was hospitalized for non-oliguric renal dysfunction. His medical history revealed 15 years of hypertension and 5 years of diabetes *mellitus* type 2. He was regularly treated and controlled with metformin, sitagliptin, ramipril and hydroclorothyazide.

A diagnosis of gastro-oesophageal junction adenocarcinoma was done 20 months before admission. The patient was submitted to pre-operative EOX (Epirubicin, Oxaliplatin and Capecitabine) chemotherapy. A total gastrectomy was made with a Roux-en-Y gastric bypass reconstruction. There was no cancer recurrence in the follow-up.

At post-surgical discharge time, 14 months before this hospitalization, the patient presented a normal renal function (creatinine of 0.8 mg/dl).

In the Emergency room, he presented creatinine of 10 mg/dl and normocytic normochromic anaemia with haemoglobin of 6.5 g/dl. Urine type II showed 30 mg/dl of proteinuria, without erythrocytes or leucocytes.

The kidney ultrasound revealed a right kidney measuring 9.9 cm and a left kidney of 8.8cm, parenchymal hyperechogenicity with cortico-medullary dedifferentiation. Serology for HIV, HCV, HBV and immunological study was negative.

Haemodialysis was immediately initiated. Three days after admission a kidney biopsy was performed.

HISTOLOGICAL FEATURES

The paraffin-embedded fragment presented a renal capsule, twelve glomeruli and some normal medium sized arteries. A frozen fragment with one glomerulus was used for immunofluorescence and was negative for the presence of IgG, IgA, IgM, C3, C1q, fibrinogen, albumin, kappa and lambda light chain.

The following figures presented the principal aspects of optical microscopy.

Figure 1 shows a predominantly interstitial nephropathy. The glomerulus is apparently ischaemic. A tubule

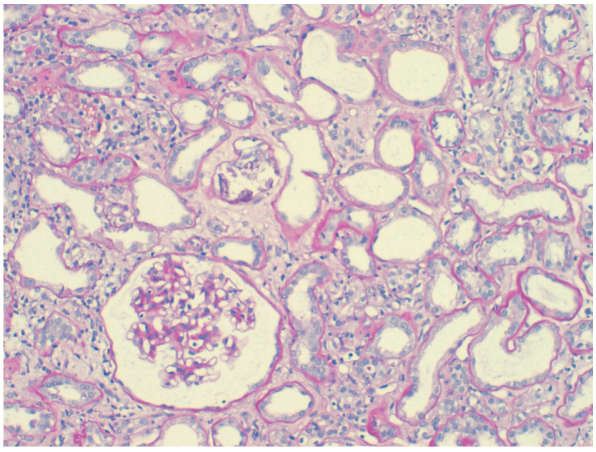

Figure 1
Periodic acid Schiff; x200.

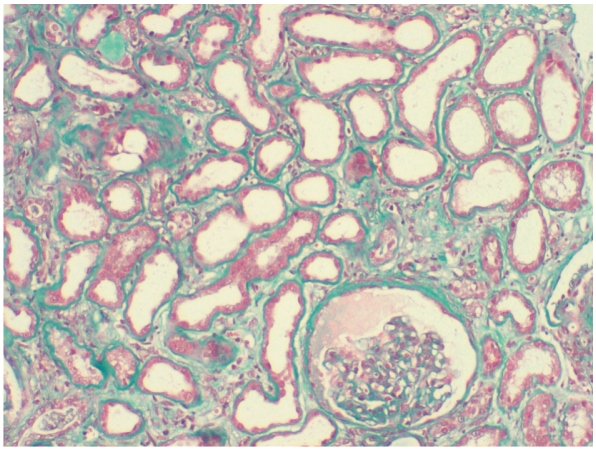

Figure 2
Masson's trichrome green; x200.

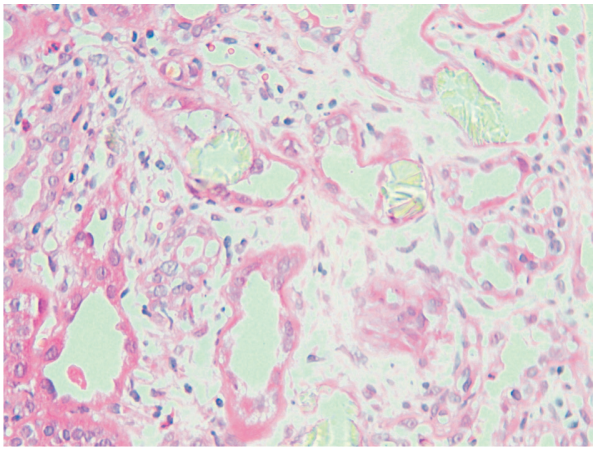

Figure 3
Haematoxylin and eosin; x400.

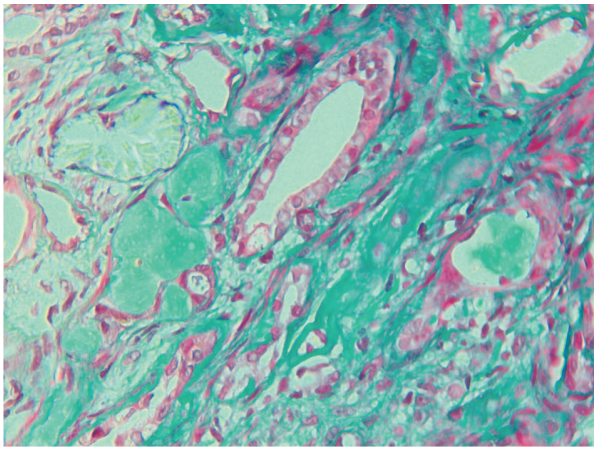

Figure 4
Masson's trichrome green; x400.

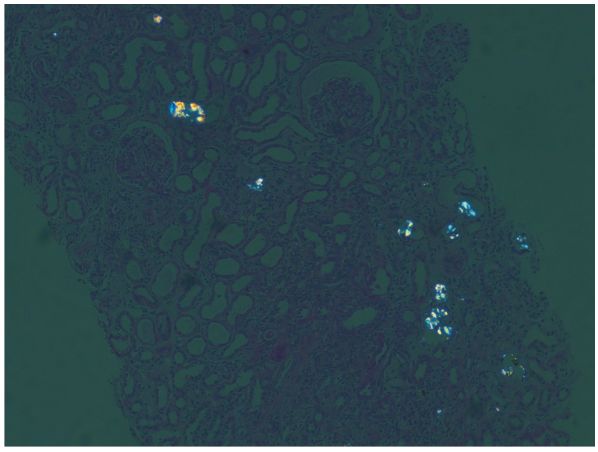

Figure 5
Haematoxylin and eosin under polarized light;x40.

presents intra-luminal abnormal substance. Figure 2 evidences acute tubular necrosis; loss of proximal tubule brush border, luminal tubular expansion and epithelial tubule cells cytoplasm simplification can be seen. The glomerulus presents ischaemic aspects. The H&E coloration in Figure 3 demonstrates several intra-luminal translucent crystals. In Figure 4, an intra-luminal translucent crystal is visible. The rest of the image shows irreversible tubular atrophy and inter-stitial fibrosis. Figure 5 presents bright intra-tubular calcium oxalate crystals in H&E under polarized light.

HISTOLOGICAL DIAGNOSIS

Oxalate nephropathy.

DISCUSSION

The biopsy showed a predominant tubulo-inter-stitial disease (Fig. 1) with some features more acute (Fig. 2) and others more chronic (Fig. 4). The

ischaemic aspects of glomeruli seem essentially secondary to the interstitial lesions and fibrosis. The glomeruli did not present parietal alteration or immune deposition. Several translucent crystals were present in tubular lumens (Figs. 3 and 4) and were brilliant under polarized light (Fig. 5), which is characteristic of calcium oxalate crystals. The calcium phosphate crystals do not change colour under polarized light.

Several studies reported that patients undergoing Roux-en-Y gastric bypass (RYGB) are at risk for hyperoxaluria, nephrolithiasis, and oxalate nephropathy[1-3]. The RYGB procedures are currently the most common bariatric operation in the United States for medically complicated obesity[1].

Nasr *et al.*[4] reported the largest series (n = 11) of oxalate nephropathy after RYGB for the treatment of either morbid obesity or gastric adenocarcinoma. The median time from RYGB to diagnosis of acute renal failure was 12 months and patients frequently had a history of baseline renal insufficiency. Clinical prognosis was poor, with 73% of patients progressing to end-stage renal disease within 3 months after diagnosis.

A study by Nordenvall *et al.*[5] in the early 80s has indicated that these patients have a normal endogenous oxalate production but have hyperabsorption of dietary oxalate. The potential mechanism for hyperoxaluria after RYGB is due to fat malabsorption and enhanced saponification of intestinal calcium[6]. Typical preventive strategies[1,2] for hyperoxaluria are prescription of a low-fat, low-oxalate diet, generous fluid intake, and use of oral oxalate binders such as calcium. Foods high in oxalate are tea, nuts, spinach, rhubarb, and cocoa.

TREATMENT AND EVOLUTION

No specific treatment was done. Recovery of renal function was not observed and this patient is in a regular haemodialysis programme.

***Conflict of interest statement*:** None declared.

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